HIV/HTLV gene nomenclature

SIR—The complexities of the genomes of human retroviruses (the human T-cell leukaemia viruses, HTLV-I and HTLV-II, and the AIDS-causing human immunodeficiency viruses, HIV-1 and HIV-2) are being unravelled at a rapid pace which is likely to continue and expand. In addition to containing a large ensemble of positive and negative regulatory genes that orchestrate virus expression, these viruses are also remarkable in that they seem to have converged onto parallel regulatory pathways. Two of the regulatory genes of the immunodeficiency viruses are analogous to the two regulatory genes of the leukaemia viruses, although their detailed mechanisms of action may be quite different. Deciphering the modes of action of the regulatory genes of these viruses is crucial to the understanding of their pathogenesis as well as to development of therapeutic agents. Because of the tremendous activity in this field, more than one name has sometimes been given to a single gene and the same name may also apply to more than one gene. In the interest of the many new investigators entering the field for the first time, we feel it is important that we reach a standard nomenclature for all known genes of HIV and HTLV. We propose the scheme outlined in the table.

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Proposed name (and derivation)	Previous names	Molecular mass ($\times 10^{-3}$)	Known function
HTLV-I and HTLV-II genes:			
tax_1 (transactivator)	x-lor, p40x, tat, tat_1, TA	41, 41, 42	Transactivator of all viral proteins
tax_2		38	
rex_1 (regulator of expression	pp27x, tel	27	Regulates expression
rex_2 virion proteins)		25	of virion proteins
HIV genes:			
tat (transactivator)	tat-3, TA	14	Transactivator of all viral proteins
rev (regulator of expression of virion proteins)	art, trs	19, 20	Regulates expression of virion proteins
vif (virion infectivity factor)	sor, A, P', Q	23	Determines virus infectivity
vpr (R)	R	?	Unknown
nef (negative factor)	3'orf, B, E', F	27	Reduces virus expression, GTP-binding
vpx (X) (only in HIV-2 and SIV)	X	16, 14	Unknown
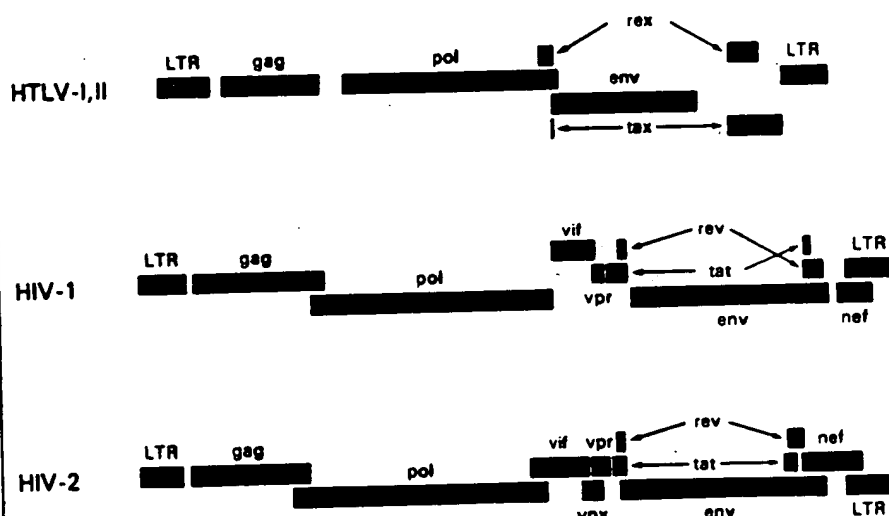
Vpr and *vpx* are temporary names and may be changed when more information about their functions is available. Subscripts 1 and 2 would be used to distinguish genes of HIV-1 and HIV-2 (for example, rev_1 and rev_2). It is expected that genes of the simian viruses (STLV-I, SIV) would follow similar nomenclature with the subscripts STLV or SIV as appropriate.

Estimating the incubation period for AIDS patients

SIR—The nonparametric analyses of the data on transfusion-related AIDS considered by Medley *et al.*[1] indicate problems of identifiability. With data obtained by retrospective determination of the time of infection for diagnosed AIDS cases, it is only possible to estimate the early part of the incubation distribution up to a constant of proportionality. The same applies to the total number of infections by blood transfusion before any given time. The transfusion data themselves are unable to discriminate between high infection rates coupled with long incubation times on the

As do Medley *et al.*[1], we postulate a function $h(x)$ which specifies the increase over time of the number of HIV-infected individuals who eventually develop AIDS, and a probability density function $f(s)$ for the incubation time of those individuals. The corresponding likelihood function can be maximized jointly with respect to h and f. As the likelihood depends only on the product of h and f, it is not possible to estimate either of these fuctions completely; they may be individually estimated only up to constants of proportionality c and c^{-1}, respectively.

nosed within t years of infection, $F(t) = \int f(u)du$, are given in the figure for the three age groups considered by Medley *et al.*. In this figure we show the estimates of $F(t)$ so that for each group, $c = F(7.5)$. For the children, the levelling of the estimate of $F(t)$ by about 3.5 years suggests that the whole of the distribution of incubation times has been seen; it may then be reasonable to suppose that $c = 1$ but, as also noted by Medley *et al.*, a second wave of incubation times that exceed 7.5 years is not excluded by these data. For the other two age groups, there is nothing in the transfusion data themselves to suggest a value for c. As a consequence, it is impos-